St. Bartholomew's Hospital Journal.

VOL. IV.—No. 38.] NOVEMBER, 1896. [PRICE SIXPENCE.

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St. Bartholomew's Hospital Journal,

NOVEMBER 14th, 1896.

"Æquam memento rebus in arduis
Servare mentem."—*Horace,* Book ii, Ode iii.

Acute Anterior Poliomyelitis in an Adult.

A Clinical Lecture delivered on October 16th, 1896,

By SAMUEL GEE, M.D., F.R.C.P.

JOHN C—, æt. 55 years, was admitted into Luke Ward, on September 21st, 1896, suffering from loss of power in the arms.

On July 25th, after a pint or two of beer he felt dizzy, went home and lay down. An hour afterwards bad headache came on. Next morning he vomited. The next three days he was delirious, especially at night; and he has no recollection of what took place during this time. For this reason our knowledge of the symptoms which attended the onset of his illness is very incomplete, and we can do no more than assume that they were the symptoms which usually occur in these cases.

In this form of spinal paralysis the onset is always sudden. The symptoms of the onset are of three classes. First, fever; we do not know that this man was feverish, but we may safely assume that he was so. Second, symptoms of an affection of the brain; in this patient, bad headache, delirium, and perhaps we may include his vomiting among the cerebral symptoms. Acute anterior poliomyelitis is very much more common in children than in adults; and in children the brain disorder often manifests itself by convulsions and by coma. Third, paralysis is an early symptom: it is sometimes marked at the very beginning of the illness; it usually attains its full extent in a few hours, or, in other words, in a few hours as many muscles are paralysed as ever will be paralysed; and, with regard to the extent of paralysis, in most cases you may say, after it has lasted a few hours, that any change will be a change for the better. As to the intensity of the paralysis, it attains its full height, sometimes equally quickly, sometimes less quickly: however, in a day or two, the degree of the paralysis will be as great as it ever will be; any change will be a change for the better. This is the rule; but as I have already said, we cannot ascertain whether the rule was followed in this man's case. It probably was not followed; probably the interval between the onset of the cerebral symptoms and of the paralysis was longer than usual, for he himself says that he did not find out that his arms were useless until a fortnight after the beginning of his illness. If this be true, for the occurrence of paralysis to be delayed so long was unusual.

There can be no doubt that the fever and cerebral disorder indicate a specific infection of the blood. In this form of paralysis there is no evidence that the brain itself is diseased. The brain symptoms,—headache, delirium, coma,—are not idiopathic, not due to disease of the brain, but sympathic; the brain is poisoned by the poisoned blood.

We may, then, safely assume that there is a specific infection of the blood; but in most cases we know nothing about

the nature of the infection. And considering this change in the blood with respect to the spinal paralysis, what shall we say? Is the blood poisoned by the disease of the spinal cord, or is the spinal cord affected secondarily in consequence of the poisoned blood? There cannot be much doubt that the blood is not infected by the cord, and for two reasons. (i) The changes in the cord are not of a kind likely to cause fever. A very small abscess will infect the blood so as to cause high fever and other very serious symptoms; but so far as we know of the changes in the cord which occur early in this spinal paralysis, there is little more than excessive vascularity and disintegration of the nerve cells and fibres. (ii) There are certain infections of the blood, which are well known (such as measles and smallpox), which are known also to cause secondary disease of the spinal cord; and disease which in all respects resembles the spinal lesions of acute anterior poliomyelitis. I myself have seen a case of acute anterior poliomyelitis dependent upon smallpox. A young man, nineteen years old, was taken ill with pain in the back and headache, on November 23rd. On November 27th the rash of smallpox appeared. On November 29th, when he awoke, both arms were completely paralysed. Some of the paralysed muscles slowly recovered, but some were left in a state of atrophy, viz. the deltoid and triceps on both sides, the right pectoralis major and left biceps. Observe that in this case the palsy did not occur till the seventh day of the disease. So that, to repeat, no such local spinal lesions are known to infect the blood, and infections of the blood are known to cause local lesions of the spinal cord. Excluding such obvious infections as smallpox and measles, in the cases of acute anterior poliomyelitis which are left, and which constitute by far the majority, the only thing about the specific infection which we know is this, that the disease is sometimes epidemic: many persons suffer about the same time in the same place. It has seemed to me to be more common in hot weather. To the best of my belief no student of bacteriology has yet endeavoured to discover the specific cause; and in order to discover it, examinations of the blood must be made, so it would seem, during the first day or two of the disease. For the symptoms of blood infection are very transitory, and seldom last longer than a few hours, or a few days, or a week at the outside. And in concluding this topic, I must confess that in some cases it is not possible to obtain a report of any symptoms indicative of blood-poisoning; the patient becomes suddenly paralysed, and that is all.

The fever and brain symptoms soon pass away in all cases, and the patient is left more or less paralysed. Here also the report which the patient gives us concerning the course of his illness is very meagre. A doctor saw him on the fourth day, and told him to keep in bed and live on slops. The patient himself tells us that he was not paralysed until more than a week afterwards, he found that he could not move his arms. But in the early stage of the disease, paralysis is easily overlooked; the fever, headache, delirium, coma, attract all the attention, and it is not observed that the patient does not move one or more limbs. So I can do no more than tell you what commonly occurs in acute anterior poliomyelitis.

The palsy at the onset is either universal or local. (i) By universal I mean that all the voluntary muscles are affected, excepting the respiratory muscles and those animated by the cranial nerves. (ii) By local palsy I mean palsy confined to one or two limbs, or to certain muscles in a limb, or even to certain portions of a muscle. Whether our patient's palsy was universal or local, at first, we do not know; it probably was local. And so we will pass on to the next fact.

On August 3rd (or on the tenth day of illness), he went into the Staines infirmary; he stayed there five weeks, and when he left, his condition was much the same as at present,—and I will now set his present state before you.

There is nothing wrong with him excepting paralysis of his arms, and the pain which he complains of in his shoulders.

As to the paralysis, some of the muscles are very much paralysed, others are less so. (i) The most paralysed muscles are the deltoid of the right arm, and the biceps, brachialis anticus, and triceps of both arms. He cannot raise the right arm to a right angle with the body. He can bend both elbows pretty strongly, but if you watch the arm when he does this, you will see that the flexion is almost entirely performed by the supinator longus. These much paralysed muscles are much wasted also, as is always the case in anterior poliomyelitis. Probably many of you know that the palsy, which is due to a lesion anywhere in the lower segment of the motor tract, always is a wasting palsy; a palsy attended by muscular atrophy, and more than this, by a degenerative muscular atrophy. The lower segments of the motor tract reach from the large multipolar cells in the anterior horns of the grey matter of the cord, along the nerves till they end in the muscles. In the form of spinal paralysis from which I believe our patient to be suffering, it is the anterior horns which are affected, and hence the name anterior poliomyelitis, as it were, anterior grey myelitis. I say it is a degenerative atrophy. How do we know this? All muscles which are paralysed waste more or less, but in many cases the atrophy is not degenerative, but is apparently due altogether to disuse, and admits of the muscle being completely restored to its normal condition. But in a degenerative atrophy the muscle is destroyed, and can be restored either with great difficulty or not at all. The best means of distinguishing these two forms of muscular atrophy during life is found by making an electrical examination of the muscles. In our patient, the right deltoid will not contract under a strong faradic or induced current: under the same current the left biceps will not contract; the right biceps and both triceps muscles contract feebly. Yet all

these muscles will contract to a feebler galvanic current than is necessary to make a healthy muscle contract. These are the most important reactions of degeneration, as they are called; they are signs of degenerative muscular atrophy,—that is to say, the affected muscles have lost their excitability to faradism, either altogether, or to a great degree, whilst to galvanism they are too excitable. The reactions of degeneration occur very early in this disease. I have known faradic contractility to be quite extinct on the fourth day. (ii) A few other muscles seem weak, especially the left deltoid; but they are not much wasted, and do not give reactions of degeneration.

He complains of pain in the shoulders when you move them, and you feel a sort of crackling at the same time. The patient had nothing the matter with his shoulders before the paralysis occurred. The same thing happens in many cases of ordinary hemiplegia. I believe that there is arthritis, that nutritive degeneration occurs in the joints just as it does in the muscles; something very much like rheumatoid arthritis sometimes sets in; and when the smaller joints—wrist and fingers—are affected they may be red and swollen.

These are all the patient's symptoms; and now I will say a few words about diagnosis, prognosis, and treatment.

Diagnosis.—Wasting palsy I have declared to be due to disease affecting some part of the lower segment of the motor tract; and in this patient I believe the part affected to be the spinal marrow. But suppose somebody were to ask, what are your reasons for setting the nerves aside?—why is it not a neuritis? The only reply to this question I can make is this—that I know no kind of neuritis which affects the nerves supplying the deltoid, biceps, and triceps of both arms and those nerves only. So that I believe our patient is suffering not from a neuritis, but from a partial myelitis of the cord about on a level with the fifth cervical nerves.

The patient complains much more of the pain in his shoulders than of the palsy. I have just given you my reasons for believing that the affection of the joints is secondary to the paralysis. But let us ask this question: Is it possible that the primary disease is acute rheumatoid arthritis, and that the palsy is secondary? I say no; because in this case, the case of wasting palsy secondary to disease of joints, the muscles paralysed are the extensors of the joints. But you will reply that in this patient the extensors of the shoulders are paralysed, namely the deltoids. True; but what about the biceps muscles? His bicipites are very much more wasted than the deltoids, and it would be an unheard-of thing for disease of the shoulder to paralyse the biceps. So that, to repeat, I believe the arthritis to be secondary.

Prognosis.—Acute poliomyelitis is not a progressive disease,—that is to say, it does not go on from bad to worse. On the contrary, the paralysis is manifested to its full extent and its full intensity from the first. It is a retrogressive disease, and tends to go from bad to better. The degree of the paralysis at the outset is no guide, because muscles which are at first completely paralysed, very often, and in the course of no great length of time, completely recover. The electrical reactions are no guide because muscles which are completely inert to faradisation may completely recover. Nay, more, muscles sometimes recover their power of contracting voluntarily while they are still dead to faradisation. I think that excessive galvanic irritability is a more valuable sign, or in other words, is a bad sign, and means that the muscle will waste. It is this wasting or muscular atrophy which affords the most trustworthy prognostic. Muscles which are very much wasted cannot recover. For this reason I fear that our patient will never recover the use of his biceps muscles, at least not of the left biceps; his deltoids are in a more hopeful state. Lastly, you may take it as a safe rule, that muscles which have not recovered after six months never will.

Treatment.—For the first few weeks of the disease the best drug to administer is belladonna. There are two reasons for advising it. First, we are told that it has the power of causing contraction of the small arteries in the spinal pia mater, and local hyperæmia probably constitutes an important part of the lesion in acute poliomyelitis. Next, some of the best recoveries I have seen have occurred to patients who were treated by belladonna early in the disease. But you know how very open to error loose experience of this kind is, and yet it is not easy to attain anything better. I myself give belladonna in full doses until the pupil is dilated, or a belladonna rash appears, or the patient's head is affected, and then I lessen the dose. I put a broad strip of belladonna plaster over that part of the spine which answers to the situation of the disease in the cord.

We are doing what we can for the final result of his disease, namely, for the muscular atrophy, but I fear it is not much that we can do. It is not to be expected that we can restore muscular fibres which are utterly degenerate and destroyed, but we may hope to improve the nutrition of any that are left. With this object we are employing assiduous massage of the wasted muscles. This is a remedy which cannot do harm, and which you may begin as early in the disease as you please. We are also galvanising the muscles. Now this is a remedy more unpleasant than massage, and not so easily carried out, because it requires that a medical man should do the galvanism. A knowledge of anatomy is required. There is an opinion current that galvanism should not be used during the first few weeks, lest it should aggravate the spinal lesion. Whether there is any truth in this opinion or not I cannot say. But about one point there is no doubt, that in parts where the muscles are nicely antagonised, say in the forearm, the hand, the leg below the knee, galvanism must be used with great circumspection, lest by improving the nutrition of the

unaffected muscles, whilst doing no good to the wasted muscles, you aggravate the deformity which is apt to ensue in the parts I have mentioned. So far as I know, massage has not this bad result. Moreover, it is much more easy to carry out massage than galvanism for a great length of time.

In conclusion, let me say that in my opinion acute anterior poliomyelitis is not a disease in itself, is not a disease by itself, or in one word is not idiopathic, but that it is an incident or accident in the course of some acute specific infection of the whole body, the universal affection being primary, and the local disease secondary.

Pathological Jottings.

By A. A. KANTHACK, M.D., Lecturer on Pathology.

II.—Chronic Inflammation.

"CHRONIC inflammation" is a term in constant use,—physicians, surgeons, and pathologists alike use it, often to denote a known and recognisable condition, equally often to have a useful peg on which to hang a less defined lesion. In my previous note (this Journal, September, 1896, p. 182) I wrote that "such a thing as a chronic inflammation does not and cannot exist." This may appear to most to be either rank heresy, or the language of exaggerated generalisation, and I therefore owe some form of an explanation, and this I propose to give in the following lines. Although, partly on account of their sketchy character, they may fail in breaking up the old belief, which in unflattering expressiveness I called archaic, I hope that they will define the standpoint from which I have viewed this matter since 1888, when in Virchow's laboratory I worked at "chronic laryngitis."

What is a chronic inflammatory process? Examples of so-called chronic inflammations are chronic nephritis, chronic peritonitis, chronic laryngitis, chronic conjunctivitis, chronic endometritis, and, *sit venia verbo*, chronic endocervicitis. Some writers, *e.g.* Ziegler, even include chronic abscesses and chronic ulcers in this category. These latter processes are, in this country at least, not regarded as chronic inflammations, and for the present I shall leave them out of consideration, especially as they form splendid subjects for subsequent "jottings."

If we look at the various forms of so-called chronic inflammatory processes, we find that histologically they may be classified under several headings. (1) In some we find a hyperplasia or proliferation of the connective tissue; or, if a mucous membrane be affected, a hyperplasia both of the epithelium and underlying tissues, in which sometimes the glands also share; (2) in others we find so-called catarrhal conditions when the lesion occurs in a secreting tissue; (3) in others again we have interstitial fibrous changes; and (4) in others a complete replacement of the primary elements by fibrous tissue.

Let us for a moment contrast a few apparently different lesions; and to make the matter simpler let us begin with chronic laryngitis (so far as it affects the vocal cords), and chronic catarrh of the mucosa of the cervix uteri. In a chronic inflammation of the vocal cords we notice chiefly (*a*) proliferation and hyperplasia of the subepithelial connective tissue, *i.e.* fibrous hyperplasia (or in more modern language, which is not satisfied without an "-osis" or an "-oma," fibrosis); (*b*) proliferation and hyperplasia of the epithelium itself, which frequently becomes horny; and (*c*) proliferation of the capillaries and vascular elements. The proliferation may be so complete and uniform as to lead to a papillomatous growth, or a pachydermia.

In a chronic cervical catarrh similarly we have (*a*) proliferation and hyperplasia of the subepithelial connective tissue; (*b*) proliferation and hyperplasia of the secreting epithelium itself, leading to dilated and elongated, or even cystic follicles, lined often by several layers of columnar epithelium; and (*c*) proliferation of the capillaries and smaller vessels. Here also the proliferation may be so complete and uniform as to lead to a beautifully papillomatous surface. The proliferated epithelium retains its secretory activity, and therefore we have the catarrhal flow. This is the only apparent difference between this affection and the laryngeal one; but here it is absent, because the squamous epithelium is not secretory in the ordinary sense of the term. The catarrhal flow, however, merely stands for increased functional activity, and that we have also in the case of the laryngeal lesion, where it shows itself in the shape of increased formation of horny substance (keratin). Hence we must allow that in point of principle there is no difference between these two processes which at first sight appear to be distinct; and therefore to the three factors mentioned, viz. hyperplasia of the connective tissue, hyperplasia of the epithelium, and slowly increasing vascularity, we must add a fourth, viz. increased functional activity. These changes are frequently, if not commonly, found in so-called chronic inflammations of mucous, muco-cutaneous, or cutaneous surfaces. But we cannot say that they are found in every instance of what is described as chronic inflammation. They are, therefore, not to be regarded as necessary criteria: excepting the fibrous changes, one or other may be absent. The "fibrosis" is an essential attribute of chronic inflammation. This will become clear, if we consider other examples.

In some cases, in place of a hyperplasia we notice, that on the contrary the mucosa is atrophic, as *e.g.* in atrophic rhinitis or gastritis. The attribute "atrophic" is, however, merely a macroscopic and not an essential distinction, for

on microscopical examination we find, during certain stages at least, firm fibrous tissue, contracting from the surface and, so to speak, smothering the glands, which for a long time remain functionally very active—*teste* the fœtid secretion of ozæna, or the cystic dilatation of the glands in atrophic gastritis. In such cases, it appears as if instead of a hyperplasia we had an induration of the connective tissue, without proliferation of the surface epithelium and the capillaries. We must, however, keep in mind that induration could not have occurred without previous proliferation, the newly-formed fibrous tissue becoming condensed as soon as it is formed: it requires a proliferative stimulus for induration or—to borrow from modern language again—for sclerosis to ensue.

To us, as more or less casual observers, viewing matters through compound microscopes, it must seem to be somewhat of a chance whether newly-formed fibrous tissue should contract or go on increasing. A scar will generally condense into hard fibrous tissue, but occasionally it will become cheloid. It appears that the connective tissue, as Grawitz would have put it, having once awakened, there is necessarily no limit to the energy of its waking hours, that is to the fibrous hyperplasia; it may go on unchecked, in a condition of morbid insomnia, to continue the metaphor; but at other times it may stop at a certain point and it may either remain there, or condense into hard or indurated tissue. It must further be remembered that atrophy and polypoid hypertrophy (in the stomach, for instance) may occur together. Again, epithelial proliferation is frequently present in atrophic "inflammations," when, for instance, we find in ozæna the stinking mucosa lined by several layers of squamous epithelium, the product of a proliferative metaplasia, and papillomatous cysts in atrophic gastritis. So that even in these conditions we may have three of the four above-mentioned conditions present, although in modified form, viz.: (1) induration of the subepithelial connective tissue; (2) partial or complete proliferation of the epithelium; and (3) increased functional activity, the increased vascularity being impossible on account of the induration.

Chronic inflammations of the serous membranes, such as the pleura, peritoneum, tunica vaginalis, or pericardium, present themselves in the form of thickening which may vary considerably in degree. We may have either mere opacity or thickening, with or without contraction, *i. e.* we have fibrous hyperplasia with or without induration. Frequently there is also distinct hyperplasia of the epithelium (or endothelium), which by a process of metaplasia may even become converted into a kind of squamous epithelium, as in a specimen of thickened peritoneum examined by myself. Increased vascularity is often present, and with this hydrops is frequently associated, which followers of Heidenhain would be inclined to regard as an increased functional activity of the endothelium. Here, then, all the four factors may be present, although one or more of them may be absent, but the fibrous changes are always there.

We now come to another form of chronic inflammation, viz. the chronic interstitial form. Examples of this we find in interstitial nephritis, cirrhosis of the liver, interstitial myositis and myocarditis. If we examine these lesions microscopically, we are struck with the marked fibrosis which has taken place,—fibrous tissue, more or less well formed, and often of exceeding firmness, surrounds the active or organic structures, whether they be kidney tubules, liver cells, or muscle fibres; the framework or secondary elements often may altogether outgrow the primary elements. The fibrous hyperplasia in these conditions, then, is well marked. Increased vascularity is often present, but may be absent in advanced stages: a hyperplasia of the epithelial tissues cannot of course take place where muscle is affected, as for instance in myocarditis or myositis; but in the interstitial forms of chronic inflammation it is generally absent also in organs which are largely epithelial in structure, as *e.g.* the kidney, liver, and pancreas. In an interstitial nephritis the renal epithelial substance becomes compressed and atrophied; the liver cells degenerate and disappear extensively in most forms of cirrhosis; and the pancreatic cells share the same fate.

I think that a consideration of the conditions which we find in myositis is instructive. (1) In muscle we may have, as a rule, as the effect of bacterial irritation, an acute interstitial inflammation, harmonising in its details with acute inflammation elsewhere. If this acute inflammation passes off without having caused serious lesion to the muscle fibres themselves, practically no permanent changes may be left behind. But if the acute injury did cause serious lesion, breaking up some of the muscle fibres or producing partial or total necrosis, then we shall find that repair is accompanied by formation of fibrous tissue: the foundation for a fibrous hyperplasia is thus laid. This newly-formed fibrous tissue, imbued with the progressive stimulus characteristic of all infant growth, will often extend beyond the original seat of lesion, between the sound muscle fibres, so that on transverse section at this stage one would find small and compressed muscle areas surrounded by rings of fibrous tissue, *i. e.* in current language "a chronic interstitial myositis." The effect of this compressing fibrous tissue is to cause further degeneration of the muscle fibres, and as these disappear more fibrous tissue appears—the "vicious circle" is established. (2) We have, however, another cause of chronic interstitial changes in muscle, viz. atrophy. If, for instance, a muscle atrophies after a central or peripheral nerve lesion, fibrous tissue often soon appears and takes the place of the muscle fibres, and we again have a chronic interstitial myositis. "Tissue degeneration, if not repaired, leads to fibrosis"—thus I expressed myself in my previous article; degenerated muscle fibres are replaced by invading and proliferating connective tissue. Professor Adami, who in

his views on chronic inflammation more or less agrees with me,—or perhaps I should say, with whom I more or less agree in his views,—has called this form of fibrosis "a replacement fibrosis"—a very useful term. It seems to me, therefore, that an important cause of progressive chronic interstitial myositis is the degeneration of the muscle fibres, which may be due (*a*) to an acute interstitial inflammation or (*b*) to myotrophic or neurotrophic lesions, and which (*c*) may be kept up by, or progress with, the appearance of the fibrous tissue.

Exactly similar conditions we find in so-called peripheral neuritis, as met with in diphtheria or lead-poisoning. As Dr. Sidney Martin and others have shown, the earliest stage in the process is a degeneration of the nerves, and this is followed by a proliferation of the connective tissue which may go on to fibrosis. "Tissue degeneration, if not repaired, leads to fibrosis." I need not allude to the well-known changes in the spinal cord; everybody knows that the degenerated tracts and areas are replaced by fibrous tissue. The law is therefore strongly supported by our knowledge of neuropathology.

If we now turn to cirrhosis of the liver, we meet first of all with considerable difficulty, since there are many different forms of cirrhosis. We have the intercellular, lobular, and biliary types, widely different and diverse in their ætiology and histology; and it seems to me impossible to explain them all in the same manner. I am not going to discuss their causation and development here, however much I am tempted to do so, but wish to mention a point which has also struck Professor Adami and others, before we began to think on the matter. It seems to me that when the cirrhosis is so far advanced as to cause so marked a degeneration of the liver cells that recovery is impossible, then the degenerate cells may act as a further stimulus for progressive fibrosis. I am not yet quite prepared to believe that the primary cause of cirrhosis is *always* a degeneration of the hepatic cells, because we frequently find advanced fatty and amyloid degenerations without fibrosis; but I do believe that, when the process of cirrhosis has once begun, the degenerated cells are replaced by fibrous tissue, and that therefore the degeneration is to some extent responsible for a continuity in the cirrhotic process. Obviously the connective tissue must be in a position to respond by proliferation, before a fibrosis can result. If it be impaired, either because the whole individual is atrophying, or because it is itself hopelessly badly nourished, fibrosis cannot possibly take place. Therefore our law only applies, if the connective tissue be relatively sound or, at any rate, capable of proliferation.

Venous engorgement occasionally, though rarely, leads to induration in various organs, *e. g.* the liver, kidney, and spleen. This is probably due to the fact that on account of the engorgement, degeneration of the organic cells has ensued, which are then replaced by proliferated connective tissue. But generally the tissues are too badly nourished to respond by proliferation. We cannot expect a complete fatty degeneration of the liver to lead to fibrosis, because generally the cause of such degeneration implies a general debility or a hopeless condition of the connective tissue, and the same applies to amyloid disease. When, however, such general debility or impairment is absent, then we find that even a complete fatty metamorphosis of the liver may be accompanied by fibrosis, *e. g.* in true fatty cirrhosis. At present I am not courageous enough to declare that ordinary cirrhosis of the liver is always due to proliferative changes in the interstitial, portal or lobular, connective tissue, appearing in response to cell degeneration, although I have a strong inclination towards such a view; I believe, however, that the degenerated cells promote the progress of the fibrosis.

Ordinary interstitial nephritis (red kidney) we may also explain as being produced by primary hyperplastic changes in the interstitial connective tissue; but it has to be proved yet that it is a primary hyperplasia, and not a hyperplasia called into existence by degenerative changes in the renal tissue. When the fibrosis becomes excessive, it no doubt causes organic destruction, which in its turn favours fibrosis. The chronic interstitial changes in a white kidney are certainly due to several factors: (*a*) the repeatedly recurring attacks of acute or subacute inflammation; (*b*) the organic destruction resulting therefrom, which awakens the connective tissue; and (*c*) the proliferative energy of the connective tissue, which I shall explain shortly.

If now, in order not to make this article too wearisome, we review shortly the various forms of chronic inflammation, we find that we have—(*a*) processes which begin primarily in the connective tissue; fibrosis appears and progresses, the process being in part maintained by the destruction of the organic elements (by Professor Adami termed *productive fibroses*); (*b*) processes which begin with an atrophy of the organic elements, the latter being replaced by hyperplastic connective tissue (by Professor Adami termed *replacement fibroses*); (*c*) processes which occurring on free surfaces involve all structures concerned, but where again the most striking phenomenon is the fibrous hyperplasia (by Professor Adami included under *productive fibroses*).

It is evident that "chronic inflammations" are different and diverse in their nature, origin and appearance.

An important law to which I am always fond of recurring is that "Tissue degeneration, if not repaired, leads to fibrosis," provided of course that the connective tissue is capable of further growth—for if it be half dead itself it cannot possibly assume fresh vigour,—and provided also that the stimulus for proliferation is sufficient, or, adopting Grawitz's metaphor, that the connective tissue has been sufficiently roused and awakened. It is curious how frequently biologists look at pathological phenomena in exactly

the opposite way to that which suggests itself to those who spend their days—and often their nights as well—in studying these phenomena. Thus W. Roux, in his work *Der Kampf der Theile*, makes an effort to persuade us that the degeneration of the nobler tissues (as Professor Adami calls them) is due to the invasion of the interstitial tissue, *i. e.* that we have a struggle (Kampf) between the highly differentiated tissues and the connective tissue; and if the latter win, degeneration and fibrosis result. Now it must be obvious to all pathologists that such a struggle does not exist, and many will agree that generally it is the degeneration of the nobler tissue which gives the connective tissue an opening. The tissues abhor atrophy and necrosis, and if homologous repair be impossible, the connective tissue fills up the gap.

Having briefly discussed some of the commoner types of chronic inflammation,—chronic endocarditis, atheroma and the various forms of endarteritis I shall leave for a future occasion,—I must now pass to the most important question, "Is chronic inflammation an inflammation?" I have already given my answer, which is as decidedly negative as it possibly can be, and to some no doubt must sound like rank heresy. I shall not attempt to define inflammation, because such an undertaking would occupy pages and pages, and because, with all due deference to those who have attempted to define it, I feel that no existing definition is absolutely satisfactory. We know inflammation by its appearances and phenomena, and we must remember that *inflammation is not synonymous with repair.*

If we look at microscopical specimens of tissues and organs in a condition of so-called chronic inflammation, we do not find the appearances of inflammation, but we find appearances characteristic rather of repair by fibrous tissue. True, at the outskirts of a chronically inflamed area we may often detect a few dilated vessels surrounded by clusters of round cells, but the bulk of the specimen shows nothing that one could call inflammation. If with Professor Adami we venture to define inflammation as a *series of changes which constitute the local attempt at repair of an injury to the part*, then chronic inflammation, exhibiting all the changes of repair, cannot possibly be inflammation, for *inflammation ceases where repair begins*, and chronic inflammation is a term which has been given to conditions which show already *completed* repair, or which show *excessive* repair. This excessive repair, to my mind, is an important element in some forms of "chronic inflammation." By excessive repair I mean the hyperplasia and hyperplastic tendency of newly-formed fibrous tissue.

An acute inflammation, in the language of the surgeon or physician, is frequently followed by a chronic inflammation. What does this signify? Merely this, that the acute process has been repaired by fibrous tissue, developed from the proliferating connective tissue, but the latter once awakened to increased growth, in the full enjoyment of renewed vigour, continues to develop further and further on the slightest provocation. An acute inflammation is often the precursor of a fibrosis, but surely that is no justification for calling the resulting fibrosis a chronic inflammation.

An acute nephritis may at once pass into gradual and progressive induration (contracting white kidney) on account of such excessive repair. But in most cases where a fibrosis has followed upon an acute inflammation we have had an injured and dying tissue left behind which acts as the proliferative stimulus upon a responsive and awakened connective tissue. And what is still more important, in most cases the acute inflammation recurs from time to time and rouses the connective tissue to continued repair, when it is already in a condition of initial fibrosis, ready to proliferate, so that every fresh acute attack only makes matters worse. A fibrosis may therefore result from a single acute inflammation or from repeated attacks of acute inflammation, but on this account it is not to be considered an inflammation.

Instead of repeated attacks of inflammation there may be repeated or continued irritation, which does not necessarily produce, or may stop short of producing, an inflammation. Thus the constant use of the voice may lead to a so-called chronic laryngitis, and from other causes the same effect may ensue. Exertion and irritation are followed by hypertrophy and hyperplasia, and in this manner we may obtain a true pachydermal condition of the vocal cords, without inflammation ever having existed. Such a state of things is at any rate conceivable, if it should be objected, that my premises are unproven by actual demonstration. In most cases, no doubt, repeated irritation does lead to repeated attacks of inflammation, so localised and so slight that they are not recognised, subjectively or objectively, but nevertheless sufficient to awaken the connective tissue to hyperplasia and fibrosis, and also to cause a hyperplasia of the epithelial elements and an increased functional activity. But even then the inflammatory attacks themselves do not constitute the chronic inflammation, they simply incite to hyperplasia and hypertrophy.

The treatment of so-called chronic laryngitis has always puzzled me. The trouble is the thickened condition of the cords, due to hyperplasia and keratinous changes of the epithelium, and proliferation of the subepithelial tissue. Yet the recognised treatment is strong irritation, produced by injecting or applying all sorts of astringent substances. No doubt this is good practice for those who wish to learn laryngological methods, but does it ever do much good? In my experience, which is not only that of an onlooker, hardly ever, not to say never. And how can it? It simply increases the fibrous and the pachydermal condition by exciting inflammation.

Similarly chronic catarrhs are generally the result of continued irritation, leading probably to slight inflammatory processes, sufficient to act as a proliferative tissue-stimulus, and to lead to a permanent change in the mucosa which cannot

be described as an inflammation, but is a hyperplasia and a hypertrophy. Cauterisation is often used to cure chronic catarrhs: of course it cures them if you only cauterise enough, to remove the whole mucous membrane, whether it be in the nose or cervix. Curetting, too, will be followed by the same success.

That a chronic peripheral neuritis is not an inflammatory lesion, I need not explain again, but I must say a few words regarding interstitial processes and especially cirrhosis of the liver and kidney. Here also we have nothing suggestive of an inflammation; we have, however, fibrosis. But it may be said, that this is the outcome of a previous inflammation. I simply answer, that I see no evidence of this, and that the processes appear in the interstitial tissue, which has been awakened either by irritant substances, actual or imaginary, or by degenerating cells, or by a combination of the two stimuli. Possibly here and there an acute hepatitis or nephritis may have existed to begin with, but then it merely acted as the initial stimulus. The essence of the cirrhosis is the progressive fibrosis, which appeared either independently of an inflammation, or in the wake of an inflammation as excessive or hyperplastic repair.

Of myositis or myocarditis I need say no more than, that by the time the process has become chronic, no trace of inflammation is visible, if it ever existed. But I may be called to book, and it may be objected, that necrobiosis and necrosis produce inflammation, and that therefore tissue degeneration leads to inflammation, and that the term chronic inflammation is justified. That is only true to a certain limited extent, where we are dealing with large necrotic areas, infarcts, hæmorrhages, and such like conditions, but is assuredly not true when we are dealing with progressive degeneration. And even infarcts and necrotic areas may disappear in a scar, without an actual or real inflammation ever having existed. Moreover, I am not discussing here the repair of such lesions as necrosis and infarcts, but only these conditions which are generally recognised or described as "chronic inflammations." We can only say that tissue degeneration is a stimulus to repair: if the tissue can be repaired by a like tissue, so much the better; if not, the connective tissue must come to the rescue. The necrosed elements must first be removed. This may be done by a process of absorption or phagocytosis—if a term ending in "-osis" be preferred—with or without inflammation. This having been removed, then the fibrous tissue fills up the gaps. If there is only one gap to fill up, we have a cicatrix or repair—but that is not chronic inflammation; if, however, the necrosis or degeneration was both extensive and progressive, and is responded to by equally progressive reparative proliferation, then fibrosis ensues, or in ordinary language chronic inflammation.

I regard, therefore, chronic inflammation as a hyperplastic change of the connective tissue, occasionally accompanied by hyperplasia and hypertrophy of the epithelial and glandular elements, produced either by repeated or continued irritation (extrinsic or intrinsic), or by single, and more often, by repeated attacks of inflammation; or called into existence by progressive tissue degeneration, when the epithelial and glandular elements, of course, do not share in the hyperplastic process. An inflammation it is not, because histologically it is a process which is solely concerned with tissue elements which we consider characteristic of repair; inflammation is not even a constant precursor. It would, therefore, be well to abolish the term chronic inflammation from morbid anatomy and histology, if not from clinical medicine and surgery; but at any rate we should make an effort to be clear in our minds what the pathology of a chronic "-itis" is, before we presume to diagnose or describe it or before we attempt to treat it.

In my next article I shall discuss the subject of "Fatty Degeneration and Infiltration."

The Nomenclature of some of the Wards.

By F. A. HOWARD CLARKE.

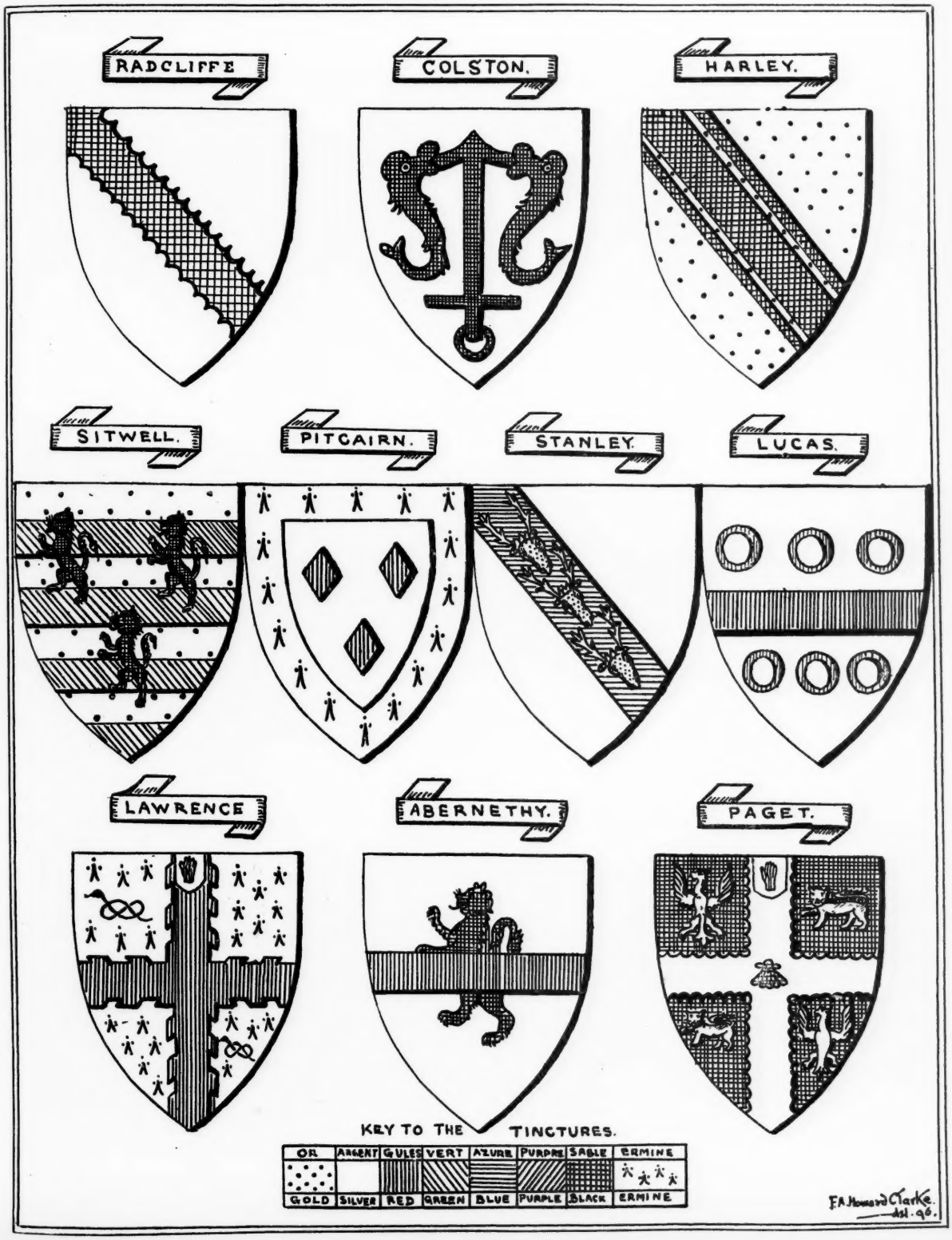
AWARD may receive the name of an individual for two reasons: first, for the purpose of differentiation; secondly, that it may be a perpetual memorial of the benevolence, professional attainments, or other claim to distinction of the individual.

A brief account is here given of some of those who have in this manner been associated with the Hospital. The armorial bearings are sketched in the hope that they may be of assistance to those artistic spirits who are interested in the Christmas decorations of the wards.

COLSTON [EDWARD]. 1636—1721. A Benefactor to the Hospital. A wealthy merchant of Bristol, he used his riches in the cause of mankind. Many almshouses and schools were founded and endowed by him, and many hospitals received large donations. He represented Bristol in Parliament from 1710 to 1713. He was buried in the church of All Saints at Bristol, and the reverence with which his memory is cherished in that town is shown by the fact that flowers are placed on his tomb every Sunday.

Arms.—Argent, between two dolphins haurient, respecting each other, an anchor, all sable.

RADCLIFFE [JOHN]. 1650—1714. A Governor of, and Benefactor to the Hospital. He was educated at the grammar schools of Wakefield and Northallerton, and subsequently entered University College, Oxford, graduating in Arts in 1669, and in Medicine in 1675. Settling first in Oxford, he moved in 1684 to London, where he soon acquired a large practice, more, it is said, by his ready wit than by his learning.


RADCLIFFE
COLSTON.
HARLEY.
SITWELL.
PITCAIRN.
STANLEY.
LUCAS.
LAWRENCE
ABERNETHY.
PAGET.
KEY TO THE TINCTURES.
OR
ARGENT
GULES
VERT
AZURE
PURPRE
SABLE
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GOLD
SILVER
RED
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BLUE
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BLACK
ERMINE
F.A. Howard Clarke. del. 96.

Among his patients were Princess Anne of Denmark, (afterwards Queen Anne), and William III and his consort Queen Mary. A baronetcy was offered to him and declined. Much of the wealth which he made by his practice was left in trust for various objects, chiefly educational. From these funds the Radcliffe Infirmary and Observatory were built, and the Radcliffe Library founded. A sum of £600 per annum was left to the Hospital.

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RAHERE. Died 1144. The Founder of the Hospital. He is first heard of as a frequenter of the court of William Rufus. Whilst on a pilgrimage to Rome he contracted malarial fever, being most probably nursed, during his illness, on the Island of St. Bartholomew in the Tiber. During convalescence he believed that he was directed by a vision of St. Bartholomew to build a hospital. It is related that in a subsequent vision the Saint appeared to him and added the building of a church to his original plan. The tomb of Rahere, which may be seen in the church of St. Bartholomew the Great, bears an ancient stone effigy of him in his habit as an Augustinian canon, and is surmounted by a much later perpendicular canopy.

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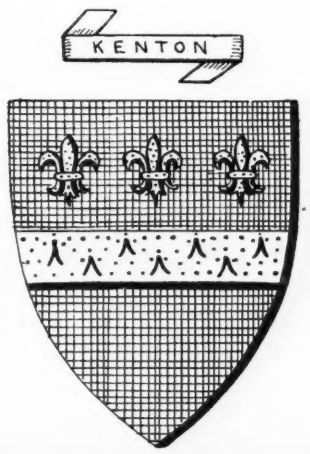

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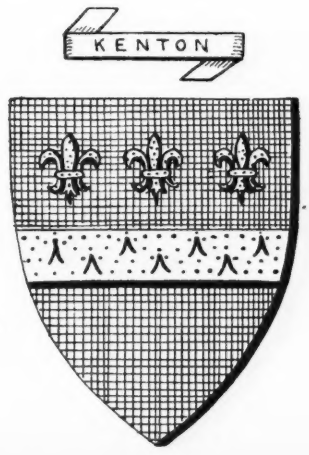

Notes.

THE ENTRY of students this year is as follows:—

Full entries	84
Special entries	59
Prel. Scientific Class	22
Total	165

* * *

AS COMPARED with last year there is a considerable fall in the number of full students, whilst entries to special classes remain about the same. The figures for 1894 and 1895 are as follows:—

	Full Entries.	Special, including Pr. Sc. Classes.	Total.
1894	119 ...	74 ...	193
1895	105 ...	82 ...	187

* * *

THE TOTAL ENTRY to the Metropolitan schools shows a considerable falling off. At Guy's there are 70 full students this year compared with 80 last year, and at St. Thomas's there are only 41 compared with 73 last year, whilst St. Mary's have 54 compared with 71 last year.

* * *

ON THE OTHER HAND, the entry in the Provinces shows only a very slight diminution, and in some cases a substantial increase; thus, at Manchester there are 74 full students, and at Liverpool 47, compared with 66 and 37 last year respectively.

* * *

AT CAMBRIDGE, 135 students have entered for the study of medicine.

* * *

DR. NORMAN MOORE has been elected a member of the Council of the Royal College of Physicians.

* * *

DR. W. S. CHURCH has been nominated by the President of the Royal College of Physicians to be a member of the Council of University College, Bristol.

* * *

A. B. WARD and H. J. MAY have taken the degrees of M.B. and B.C. of the University of Cambridge. Mr. Ward's thesis was on the "Etiology of Chorea," and Mr. May's on "Diphtheria as treated by Antitoxin."

* * *

MR. H. H. R. SKEY has passed first in the Naval Medical Service, at the examination recently held. He obtained 2860 marks. Mr. F. H. Nimmo passed eighth at the same examination with 2224 marks.

* * *

DR. J. H. DRYSDALE has been elected Assistant Demonstrator of Physiology *vice* Dr. Bowman, deceased.

* * *

DR. L. E. SHORE has been appointed an Examiner for the Second M.B. Examination at Cambridge.

THE number of names sent in for reproductions of Ouless' portrait of the late Mr. Mark Morris has nearly reached 100—unless the number reaches 200 the reproduction of the portrait will not be undertaken. Those who would like to have copies, at a cost of about 10*s.* 6*d.*, are asked to send in their names to the "Manager of the JOURNAL."

Amalgamated Clubs.

NEW MEMBERS.

A. F. C. Pollard.	F. G. de G. Best.
O. Inchley.	J. A. Lloyd.
A. Goodall.	E. L. Martin.
G. S. Ewen.	H. H. Butcher.
E. Talbot.	H. M. Huggins.
M. H. M. Melhuish.	H. Falk.
A. E. Lord.	W. Burnand.
L. Orton.	W. R. Read.
H. J. Slade.	F. H. Noke.
F. Coleman.	G. Browse.
F. A. Rose.	N. Macfadyen.
F. Bodvet-Roberts.	C. L. C. Owen.
E. C. Williams.	V. G. Ward.
R. Holtby.	A. E. Thomas.
R. T. Worthington.	V. Monckton.
W. D. Harmer.	E. P. Sewell.
E. H. Scholefield.	C. J. Thomas.
C. N. Davis.	R. L. Thornley.
P. A. Ross.	E. C. Mackay.
H. F. Parker.	E. S. Ellis.
S. D. Gerrish.	T. C. Neville.

GOLF MATCH.

GUY'S HOSPITAL *v.* ST. BARTHOLOMEW'S HOSPITAL.

On October 21st the third annual golf match between Bart.'s and Guy's was played at the Stanmore Golf Club ground. It was a beautifully fine afternoon, and the links were in excellent condition, considering the recent heavy rains, the putting greens being specially fast. Bart.'s won the first match, in 1894, at Stanmore, by 7 holes. Guy's won the second match at Bickley, in 1895, by 14 holes, Bart.'s winning the present match, as will be seen below, by 25 holes. Undoubtedly the stronger team won, two men in the Guy's team being almost novices at the game. Details of the play:—

BART.'S.		GUY'S.	
1. Mr. F. Robertson	3	Mr. C. Coventry	0
2. ,, W. D. Harmer	0	,, J. Bevis	1
3. ,, P. Furnivall.....	9	,, G. Duncan	0
4. ,, E. L. Evans	0	,, F. G. Thomas	4
5. ,, W. Whitwell	2	,, C. Shepherd	0
6. ,, A. N. Weir	0	,, A. W. Soper	2
7. ,, R. C. Bailey	9	,, C. Gray.....	0
8. ,, W. Amsden	9	,, A. C. Lacey	0
	32		7

1. Both men were short of practice, Mr. Coventry just having returned from a sea voyage; but Mr. Robertson held the upper hand all through, winning by 3 holes.

2. Mr. Harmer, when 5 up and 6 to play, went to pieces, and lost the last 6 holes.

3. A better match than the result would indicate. Mr. Furnivall playing his best game, won somewhat easily.

4. This was an amusing match; rumour hath it that several holes were decided by spinning a coin. Mr. Evans was suffering from want of practice.

5. A ding-dong fight all the way, resulting in a win for Mr. Whitwell by 2 holes.

6. A very fine match; all square at the turn, the winning ball on the way home holing out in 36.

7 and 8. These matches need no description, Mr. Gray and Mr. Lacey being no match for their respective opponents.

STAFF *v.* STUDENTS.

This annual fixture was played at Mitcham on June 30th. A strong wind somewhat interfered with the "long game" of several members of both teams; but it was a fine afternoon, and the green was in very good condition. The Students again asserted their superiority at the game of golf, this being their third victory, the Staff claiming a solitary win in 1895. Details of the play:—

STAFF.		STUDENTS.	
1. Mr. T. Smith	0	Mr. F. W. Robertson	6
2. „ H. Marsh	0	„ H. W. Lance	0
3. „ P. Furnivall	4	„ W. D. Harmer	0
4. „ R. C. Bailey	5	„ W. Whitwell	0
5. „ A. N. Weir	0	„ Ellacombe	3
6. Dr. Calvert	0	„ E. L. Evans	5
7. Mr. A. Bowlby	0	„ W. Amsden	2
	9		16

ASSOCIATION FOOTBALL CLUB.

RESULTS OF MATCHES.

Oct. 6 ...	*v.* *Forest School ...	at Walthamstow ...	drn. ...	2—2
Oct. 10 ...	*v.* Crouch End ...	at Wood Green ...	lost ...	3—4
	v. *Norseman F. C. ...	at Winchmore Hill...	won ...	4—3
Oct. 17 ...	*v.* Barnes	at Winchmore Hill...	won ...	3—1
Oct. 21 ...	*v.* *Felstead School ...	at Felstead	lost ...	0—7
Oct. 24 ...	*v.* Reigate Priory ...	at Reigate	drn. ...	2—2
Oct. 28 ...	*v.* *St. Mary's Hosp. II	at Winchmore Hill...	won ...	1—0
Oct. 31 ...	*v.* *Aldenham School	at Aldenham ...	won ...	6—2
Nov. 4 ...	*v.* Hastings Athletic	at Hastings	drn. ...	3—3
Nov. 7 ...	*v.* Old Brightonians	at Winchmore Hill...	won ...	9—2
	v. *St. John's College	at Oxford	drn. ...	3—3

* Reserve matches.

ST. BART.'S HOSPITAL *v.* CROUCH END.

This match, which was played at Wood Green on October 10th, resulted in a win for Crouch End by 4 goals to 3. The first part of the game was marked by a want of combination on both sides. Crouch End, however, soon got better together, and quickly scored 2 goals. Bart.'s then got together a little better, and Woodbridge scored from a pass by Joy. Before half-time both teams again scored, Pickering rushing the ball through our opponents' goal, the score then being Crouch End 3, Bart.'s 2. In the second half the game became a good deal faster and more combined. For a time the Hospital pressed, and Pickering again scored. Crouch End, however, replied, and the game resulted as above.

Team.—E. P. Court (goal), R. P. Brown, L. E. Whitaker (backs), A. H. Bostock, N. H. Joy, D. S. Gerrish (half-backs), T. H. Talbot, C. A. Robinson (right wing), E. W. Woodbridge (centre), H. J. Pickering, H. N. Marrett (left wing), (forwards).

After the match, Mr. Gamage and the directors of the Wood Green ground kindly entertained the teams to tea and a smoking concert.

ST. BART.'S HOSPITAL *v.* BARNES.

This match was played in wretched weather at Winchmore Hill, and resulted in a win for the Hospital by 3 goals to 1. Bart.'s won the toss, and Barnes kicked off from the pavilion end. The state of the weather rendered accurate play impossible. During the first half-time Bart.'s scored twice through Willett and Marrett. In the second half Barnes scored, but the Hospital quickly getting together again once more led by 2 goals, thus winning as stated above.

Team.—E. P. Court (goal), R. P. Brown (captain) and L. E. Whitaker (backs), A. H. Bostock, D. S. Gerrish, and H. J. Pickering (half-backs), T. H. Talbot and C. A. Robinson (right wing), J. A. Willett (centre), E. W. Woodbridge and H. N. Marrett (left wing), (forwards).

ST. BART.'S HOSPITAL *v.* REIGATE PRIORY.

This match was played at Reigate on October 24th. Reigate won the toss and elected to play uphill. The play from start to finish was fast and exciting, and at times rather vigorous. For the first twenty minutes the game was fairly level, Bart.'s having rather the best of it. At last a capital run by Talbot and Robinson on the right took the ball to the Reigate goal, and from a pass by Robinson Woodbridge scored. Six minutes later Robinson added to the Hospital score. Just before half-time Reigate scored with a capital shot, which Langton could not reach. On resuming, Reigate attacked vigorously, but failed to score till just on time, the game thus ending in a draw of 2 goals each.

Team.—J. M. Langton (goal), R. P. Brown (capt.), M. G. Winder (back), A. H. Bostock, D. S. Gerrish, H. J. Pickering (half-backs), T. H. Talbot, C. A. Robinson (right wing), J. A. Willett (centre), E. W. Woodbridge, H. N. Marrett (left wing), (forwards).

ST. BART.'S HOSPITAL *v.* HASTINGS ATHLETIC.

This annual match was played at Hastings on November 4th before a good attendance. Hastings had their strongest side, while the Hospital were handicapped by the absence of Whitaker and Bostock. To add to their misfortunes R. P. Brown missed the train and arrived twenty minutes late. Hastings won the toss, and at once attacked and quickly scored three times. The Hospital now woke up and commenced a vigorous attack, which resulted in Woodbridge scoring rather luckily off an opposing player. The teams crossed over with the score 3—1 in favour of the home team. On resuming Bart.'s kept up the pressure and Willett rushed the ball through the goal, but the point was disallowed for handling. Soon after, however, Pickering scored from half-backs, and just before the close Willett equalised. A good game resulting in a draw of 3 goals each.

Team.—J. M. Langton (goal), R. P. Brown, L. Orton (backs), M. G. Winder, D. S. Gerrish, H. J. Pickering (half-backs), T. H. Talbot, C. A. Robinson (right wing), J. A. Willett (centre), E. W. Woodbridge, H. N. Marrett (left wing), (forwards).

ST. BART.'S HOSPITAL *v.* OLD BRIGHTONIANS.

This match was played on November 7th, at Winchmore Hill. Old Brightonians brought down a weak team, and were, in consequence, severely beaten. Bart.'s having lost the toss, kicked off towards the pavilion goal. The Hospital immediately pressed, and in the first half scored 5 goals (Woodbridge 3, Pickering, and Willett). In the second half the Hospital quickly scored 2 more goals through Robinson and Willett. Old Brightonians then played up better and scored 2 goals. The Hospital replied with 2 more (Robinson and Talbot), thus winning easily by 9—2.

Team.—A. Goodall (goal); R. P. Brown (captain) and L. Orton (backs), A. H. Bostock, D. S. Gerrish, and H. J. Pickering (half-backs), T. H. Talbot and C. A. Robinson (right wing), J. A. Willett (centre), E. W. Woodbridge and H. N. Marrett (left wing), (forwards).

Our match with the Hastings Athletic Football Club is always looked upon as one of the best matches of the season, and this year's match proved no exception to the rule. Four seasons ago, when we went down for the first time to Hastings, we won easily, but each year they have been gradually diminishing the number of goals, so that this year we had to be content by making a draw of three goals each after a very hard and exciting game.

After the match the Bart.'s team were again very hospitably entertained by the Old Bart.'s men now in practice in Hastings. Our hosts were Mr. C. Christopherson, Mr. C. A. Coventon, Mr. C. B. Gabb, Mr. L. Jones, Dr. Trollope, Mr. T. H. Wadd, and Dr. A. Scarlyn Wilson. About a hundred invitations were issued to prominent men in Hastings, including the Mayor (Major Weston), Rev. P. F. P. Durnford, Dr. Redmayne, Mr. J. D. Hersey, Mr. N. Ballard, Mr. H. J. Holyoake, &c., and the members of the Hastings Athletic F.C.

Dr. A. Scarlyn Wilson took the chair. After a most enjoyable high tea, a very neatly arranged programme of songs had been prepared to fill in the rest of the time before catching our train back to town.

After an excellent quartette and violin solo had been rendered, the chairman, Dr. A. Scarlyn Wilson, proposed "Success to the St. Bartholomew's Hospital Football Club." He alluded to the short time there was at our disposal, and therefore he must somewhat curtail his speech. But there was a tradition at this annual tea—and they could not put the traditions aside,—and that was that this tea was always associated with its old friend "toast." He was, therefore, privileged as chairman to propose the health of the St. Bartholomew's Hospital team, which that afternoon had met the Hastings team. There was a popular idea years ago that medical students were cheerful and somewhat loud individuals, who passed their days in the dissecting-room engaged in eating pork pies and drinking stout, and in the evenings by disturbing other people's knockers. He could not say that when he joined the Hospital this was the case. Dr. Trollope, who was at the Hospital some years before Dr. Scarlyn Wilson, also denied that this was the case in his time. But whatever might have been the case, they had developed other ways of spending time and letting off steam, and he could not imagine a better way than by coming down to Hastings and playing a good game of football. They of St. Bartholomew's Hospital were proud of the fact that they belonged to the most ancient hospital in the country, and one that was not only the most ancient but the

most illustrious. They were proud of their connection with it, and they were glad in any way to keep up that connection and to strengthen the bond of union which bound them to their old Hospital. A football field was the foundation of many a fast friendship, and was a highway to health. He asked them to drink success to the Bart.'s team, coupling with it the name of the Bart.'s captain.

After a song had been well sung by Mr. J. Valerie, Mr. R. P. Brown replied for the Hospital team, thanking the Old Bart.'s men of Hastings for the very kind way in which they had entertained the Bart.'s team. The chairman then proposed the toast of the "Hastings Football Club," to which Mr. Hall replied.

Mr. B. Middleditch submitted the toast of "Our Hosts," pointing out the increasing keenness of footballers in Hastings, and this was partly due to the interest which people of the town—especially the Old Bart.'s men—took in the welfare of the club. The chairman briefly thanked the gathering for the kind way in which they had received the toast, and after some more music a very pleasant time was brought to a close.

Dr. C. B. Gabb was the first to begin the very pleasant evenings the Bart.'s team have after their match with the Hastings club. Now all the other Old Bart.'s men in Hastings have joined him, but Dr. Gabb, we believe, is the active agent in arranging the whole programme. Every year he makes a point of coming up to the Hospital and arranging matters with the captain and secretary of the Association Club, in order that as much enjoyment as possible may be got in the short time at our disposal after the match.

MATCHES FOR NOVEMBER-DECEMBER.

Sat.,	Nov.	14,—Eastbourne	Eastbourne.
,,	,,	*14,—Old Foresters II	Winchmore Hill.
Wed.	,,	*18,—City of London School	Winchmore Hill.
Sat.	,,	21,—Ealing	Ealing.
,,	,,	*21,—Ealing Reserves	Winchmore Hill.
Wed.	,,	25,—Casuals	Winchmore Hill.
,,	,,	*25,—Guy's Hospital II	Honor Oak.
Sat.	,,	28,—Ipswich	Ipswich.
,,	,,	*28,—Barnes Incognito	Barnes.
Wed.,	Dec.	2,—Enfield	Enfield.
,,	,,	*2,—Royal School of Science	Away.
Sat.	,,	5,—Newbury	Newbury.
,,	,,	*5,—Tonbridge	Tonbridge.
Wed.	,,	*9,—St. Anne's Heath F.C.	Virginia Water.
Sat.	,,	12,—Marlow	Marlow.
Wed.	,,	*16,—Berkhamsted School	Berkhamsted.
Sat.	,,	*19,—Drayton F.C.	Ealing.

* Reserves.

United Hospitals A.F.C.

The following team has been selected to play on Wednesday, November 18th, against Middlesex County at Wood Green:

Goal	...	A. E. Harrison (St. Thomas's).
Backs	...	R. P. Brown (capt.) (St. Bart.'s).
		J. Sharples (St. Mary's).
Half-backs	...	E. F. Buzzard (St. Thomas's).
		F. F. Lobb (St. Mary's).
		H. J. Pickering (St. Bart.'s).
Right wing	...	W. H. Agar (University).
		J. F. Fernie (St. Bart.'s).
Centre	...	G. P. Wilson (London).
Left wing	...	E. Ellery (Middlesex).
		A. Hay (St. Bart.'s).
Linesman	...	Mr. J. F. Walker (London).

Guild of St. Barnabas for Medical Students.

OCTOBER SESSION, 1896.

A Meeting of the Guild will take place at St. John's, Red Lion Square, Holborn, on the following date—

December 2nd.

Tea at the Mission House, Fisher Street, at 5.45; Guild Office, 6.15.

All information concerning the Guild may be had on application to the Secretary, Mr. W. T. STORRS, 14, Berkeley Road, Crouch End, N.

The Rahere Lodge, No. 2546.

AN Emergency Meeting of this Lodge was held at Frascati's Restaurant on Tuesday, November 10th, 1896; W. Bro. Godson, acting for W. Bro. Alfred Cooper, W.M., in the Chair. Bros. Cross, Willett, and Valèrie were made Master Masons, and Bros. Keetley, Barron, and Nall were admitted to the second degree. Fifty members and visitors were present, of whom forty afterwards dined together.

Smoking Concert of the Bart.'s Half-Company of the V.M.S.C.

THE members of the Bart.'s half-company of the Volunteer Medical Staff Corps assembled in force at the Champion Hotel, Aldersgate Street, on the 3rd inst., to hold their annual invitation smoker. The chair was taken by Surgeon-Lieutenant Miles, who was supported by Surgeon-Lieutenant Whayte, Captain André, Mr. J. S. Sloane, and the non-commissioned officers of the company. The guests included members of the Artists, London Irish, Civil Service, and other volunteer corps.

A capital programme had been arranged, and after Mr. Edgar had opened the proceedings with a selection of "Hymns," ancient and modern, Private Glaze delighted his hearers with a song, "The Ladies." Mr. Ben Nathan next gave a capital character study, in which he described how a jockey tried to lose a race for a monetary consideration, but was prevented from doing so by the fact that the other jockeys had been similarly commissioned. The encore was so unanimous that Mr. Nathan was compelled to oblige again, and his imitations of the various attempts of an Irishman, a dude, a Scotchman, and a Frenchman respectively to recite Tennyson's famous "Charge," brought forth a perfect storm of cheering.

Mr. Fred Smith gave "A Simple Maiden," from the "Shop Girl," and this was so appreciated that the audience compelled him to sing again. Mr. Cliff Ryland, the well-known patter vocalist and comedian, sang "The man of eccentric notions." His humorous interludes were much enjoyed.

The succeeding "turn" was one of the most enjoyable items of the evening. Mr. Herbert Lynwood is an artist of the first rank, and his rendering of the "Snowy-breasted Pearl" and "Sing me songs of Araby," were most artistically delivered. Mr. George Kenway gave some excellent "imitations," including a song as heard from a phonograph.

Mr. Avolo's fine bass voice was next heard to advantage in a song with the not inappropriate title of "Good Old English Beer." This melody must have considerably increased mine host of the Champion's exchequer, judging from the manner in which the audience emptied and refilled their tankards.

Further clever "imitations," and a well-sung song, "Gallery and Boxes," were given by Mr. Harold.

The next performer was Corporal Meade, of "Ours," whose contribution was undoubtedly the hit of the evening. It is common knowledge that the Bart.'s Company at Aldershot was very conspicuous during the last campaign. The events were very cleverly recounted to the tune of "Killaloo," and Corporal Meade was not allowed to resume his seat until the song had been heard a second time.

The senior non-commissioned officer then referred to the gain to the Company by the accession to the ranks of Surgeon-Lieutenant Miles, and he called upon the members to welcome their new officer. The welcome was a royal one, and when Surgeon-Lieutenant Miles rose to respond he was accorded a second volley. In thanking them for their kind reception, he was glad to say that he was not a stranger to No. 3. He had served in its ranks for five years, and it was a pleasure for him to resume his connection with the Company in his new position. He was sorry to find so few St. Thomas's men in the Company. He hoped that the fact of several representatives being present from that hospital to-night would be the means of recruiting from that quarter. He wanted No. 3 to work with him to obtain the Shield. Without detracting in any way the merits of the Company which now held it, he thought No. 3 was good enough and strong

enough to compete for it successfully if the members would make a special effort (loud cheers).

After this pleasant interval, Corporal Meade again took the floor, and soothed the listeners with "Sister Mary Jane's Top Note."

Several other equally enjoyable turns followed, and the evening was successfully terminated by the Secretary gathering his famous No. 3 choir together, and singing their notorious "War March and Popular Chants."

Cases of Special Interest.

Mark, bed 10.—Rheumatoid arthritis.
" " 24.—Morbus cordis.
Luke " 10.—Multiple neuritis.
" " 19.—Wryneck.
" " 22.—Pernicious anæmia.
Matthew, bed 3.—Gout, tophi in ears.
" " 5.—Mitral stenosis.
Colston, bed 2.—Rheumatism, morbus cordis, subcutaneous nodules.
" " 3.—Morbus cordis.
" " 20.—Double empyema (recovering).
Rahere, bed 10.—Diphtheritic paralysis.
" " 16.—Morbus cordis.
Faith, bed 5.—Malignant disease of rectum.
" " 15.—Morbus cordis.
Hope " 20.—Alcoholic neuritis.
" " 7.—Cirrhosis hepatis.
Mary " 9.—Abdominal new growth.
" " 10.—Jaundice.
" " 5.—Diabetes.
John " 5.—Paroxysmal heart hurry.

Appointments.

ADAMS, P. E., M.R.C.S., L.R.C.P., appointed House Surgeon to the Dorset County Hospital.

* * *

BRODIE, W. H., M.D.Edin., F.R.C.S.Eng., D.P.H., appointed Medical Officer to the First and Second Sanitary Districts of the Battle Union.

* * *

GLOVER, LEWIS, M.D.Cantab., appointed Medical Officer to the Out-patients at the Hampstead Hospital.

* * *

FARMER, W. H., M.R.C.S., L.R.C.P., appointed House Surgeon to the Royal Hospital, Portsmouth.

* * *

BRIGGS, J. H. D., M.B.Lond., M.R.C.S., L.R.C.P., appointed Assistant House Surgeon to the South Devon and East Cornwall Hospital.

* * *

HEDGES, J. H., M.R.C.S., L.S.A., re-appointed Medical Officer of Health for the Urban District of Leighton Buzzard.

* * *

WAYLEN, G. S. A., L.R.C.P.Lond., M.R.C.S.Eng., re-appointed Medical Officer of Health to Devizes Rural District Council.

* * *

CALVERT, CRACE, M.R.C.S., L.R.C.P., has been appointed House Physician to the Royal Free Hospital.

* * *

DUNN, P. H., L.R.C.P.Lond., M.R.C.S., has been appointed Medical Officer for the Fourth Sanitary District of the Hitchin Union.

* * *

VALENTINE, T. H. A., L.R.C.P.Lond., M.R.C.S., D.P.H., has been appointed Public Vaccinator for the District of Waitara, New Zealand.

Examinations.

FIRST CONJOINT.—*Chemistry and Physics.*—H. E. G. Boyle. *Pharmacy.*—G. B. D. Adams, A. H. Brewer, A. B. Brown, J. K. S. Fleming, A. F. Page. *Biology.*—F. J. C. Jeffcock, C. T. Price.

SECOND CONJOINT.—*Physiology.*—V. S. A. Bell. *New Regulations —Anatomy and Physiology.*—W. F. Bennett, H. Bond, W. C. Douglass, H. Goodman, W. G. Hamilton, S. Mason, H. G. Pinker, A. B. Pugh, C. C. B. Thompson, A. J. W. Wells, C. C. C. K. White, H. G. Wood-Hill. *Materia Medica—Old Regulations.*—W. H. Crossley, A. W. Wilkinson.

* * *

FINAL M.R.C.S. and L.R.C.P.—The following, having passed in all the subjects of the final examination, have been admitted to the above Diplomas:—W. Smith, G. E. Gardner, W. H. Roache, D. G. Drake, S. L. Box, J. Boyan, F. E. Price, C. R. Maitland, E. L. Davey, A. E. Naish, L. K. Harrison, F. H. Nimmo, H. Weeks, R. F. Baird, H. Williamson, T. H. Molesworth, H. W. P. Young, G. R. Baker, D. B. Keown, D. H. F. Cowm, J. L. Maxwell, J. F. Fernie, R. Armitage, L. F. Marks, G. S. Pownall, R. H. Nesham, E. G. Deck, C. F. Gordon, M. Blieden.

* * *

CONJOINT BOARD.—OLD REGULATIONS.—*Materia Medica.*—W. H. Crossley, A. W. Wilkinson, P. Wood. *Physiology.*—F. S. A. Bell.

* * *

FIVE YEARS' REGULATIONS.—*Chemistry and Physics.*—H. E. G. Royle. *Practical Pharmacy.*—E. B. D. Adams, A. H. Brewer, A. B. Brown, J. K. S. Fleming, A. F. Page. *Elementary Biology.*—F. J. C. Jeffcock, C. T. Price. *Anatomy and Physiology.*—W. F. Bennett, H. Bond, W. C. Douglas, H. Goodman, W. G. Hamilton, S. Mason, H. G. Pinker, H. B. Pugh, C. C. B. Thompson, A. J. W. Wells, C. C. C. K. White, H. G. Wood-Hill.

* * *

UNIVERSITY OF CAMBRIDGE.—*D. P. H. Examination.*—W. W. Kennedy, C. Todd.

* * *

UNIVERSITY OF DURHAM.—*M. B. and B. S. Examinations.*—W. J. Codrington.

Correspondence.

To the Editor of St. Bartholomew's Hospital Journal.

NURSING NEWS IN THE JOURNAL.

DEAR SIR,—The Bart.'s JOURNAL is widely read and with much interest among the nursing section of the community, both past as well as present. Could you see your way towards giving us news of each other with regard to appointments obtained by sisters and nurses, both within the hospital and after leaving it? If the Nurses' Press List or the name of the Gold Medallist were also published, it would lend an additional interest. If you could see your way to giving us a few items of such information, I do not think it would be trespassing much upon your valuable space, and I am sure would be much appreciated at home and abroad.

Believe me, yours truly,
ROSE S. WALTER.

282, Lytham Road, South Shore, Blackpool;
October 31st, 1896.

To the Editor of St. Bartholomew's Hospital Journal.

THE ABERNETHIAN SOCIETY.

DEAR SIR,—"A Grumbler" asks several questions in your last issue; we will endeavour to answer them *seriatim*.

He asks what has happened to the society lately. The society is in a flourishing state, and has a larger average attendance at its meetings than ever. As to the copies of *Punch*, it is true that as during August and September fewer members are in residence, fewer copies are taken in. In the last week of September some members kindly put the copy of *Punch* under the cushion of the presidential chair, whence, after a diligent search, it was disinterred. On October 1st the copies arrived late, and thus were not distributed with the other papers. When they were brought the newsboy deposited them in the artistic manner mentioned, but they were placed in the cases when the attendant made his next round.

The cases are kept to put the papers in, your Grumbler may be glad to learn. If he could induce other members to realise this he would be doing an excellent service. All notices to that effect have hitherto been in vain. While members will persist in removing the papers from the cases, or in taking them from one room to another,

or in using them as mats, confusion must occur, despite the best endeavours of the attendant. As an example of the way the papers and magazines are treated, we may state that the current number of the *Pall Mall Magazine* disappeared for a few days, and was then returned in a mutilated condition.

As to the sale of papers, Grumbler spoils his own case by dating his letter October 1st. The rule provides that a sale shall take place in October. Even a Grumbler will admit it is better to wait till the men have returned before selling the papers. The sale took place in due course on October 14th, and, strange to say, the name of a Grumbler does not occur in the list of purchasers.—We are, dear Sir, the much-abused HON. SECRETARIES.

To the Editor of St. Bartholomew's Hospital Journal.

SISTER MAGDALEN FUND.

DEAR SIR,—I am glad to be able to inform you that the appeal made through your columns on behalf of Mrs. Boyce (late Sister Magdalen) has been very generously responded to, as the following list will show. Mrs. Boyce again asks me to thank all her friends through you for their kind assistance to her.—Yours faithfully,

November, 1896. EDGAR WILLETT.

	£	s.	d.
Amount already acknowledged	31	5	0
Howard Marsh, Esq...	3	3	0
"L. W. A."	2	2	0
Per Dr. C. P. White...	1	10	0
Dr. T. W. Shore	1	1	0
Dr. G. C. Tayler	1	1	0
Dr. H. P. Tayler	1	1	0
Dr. Lenton Heath.....	0	10	6
H. B. Tait, Esq.	0	10	6
Miss Rolleston (Sister Elizabeth)	0	10	0
Dr. W. B. Addison ...	0	10	0
William Odell, Esq....	0	10	0
Dr. A. E. Wynter.....	0	10	0
Dr. H. Willoughby Gardner	0	7	6
Effie, Gladys, and Vawdray Capon ...	0	5	0
Miss Julia Hurlston ...	0	5	0
Total.....	£45	1	6

FOR the sake of "An Old Bart.'s Man" we must again state that anonymous letters are not considered.

Review.

SECTION CUTTING AND STAINING: a Practical Introduction to Histological Methods for Students and Practitioners. By W. S. COLMAN, M.D., M.R.C.P. Second Edition (enlarged and in most part rewritten). London: H. K. Lewis, 1896.

This is a useful little book, compiled by one who is a practical worker. It cannot, of course, be compared with such works as v. Kahlden (translated by Dr. H. M. Fletcher), or Dr. Sims Woodhead's *Practical Pathology;* but it is handier and more suited to the beginner, who is apt to be misled and confused by having to choose between a number of methods. There are but few mistakes in the book, and, generally speaking, the various hints and directions given are as good and sound as they can be. The section lifter still finds a place in Dr. Colman's laboratory; it is a barbarous instrument, and a cigarette paper which everybody carries in his waistcoat pocket is much more useful, as all who have worked in our own Pathological Laboratory know from practical experience. Dr. Colman is unduly severe on Leitz's microscopes, which we use ourselves, and which, on account of their cheapness and good quality, we do not hesitate to recommend. Leitz, who, by the way, hails from Wetzlar, although Dr. Colman refuses to place him among the first-class makers, now sells a splendid all-round microscope at £11, consisting of a non-inclinable stand with rack and pinion, substage condenser and iron diaphragm, double nose-piece, objectives 3, 7, and $\frac{1}{10}$ oil immersion and two oculars; so that there can no longer be any excuse for not possessing a useful microscope. Hot water might have been mentioned among the hardening reagents, for immersion in boiling water is one of the best, quickest and cheapest methods of hardening specimens, and they freeze and cut beautifully after having been fixed in this manner. Specimens are often sent up from a distance insufficiently hardened or fixed: hot water is preferable to weak spirit or whisky. The hardening properties of Müller's fluid can be considerably increased by warmth, spirit, or formalin, as described on page 20, but it is erroneous to imagine that it requires from four to eight weeks to harden in Müller's fluid; in most cases, for quick and withal good work, a week is enough. Dr. Colman gives but meagre instruction as to the preparation of specimens in the shortest possible time, and yet this is, perhaps, the most important branch of morbid histology. With the hot water method a specimen can be cut and stained in less than fifteen minutes, with the formalin method in less than one hour, with the paraffin method in less than four hours, and with the freezing method by an experienced worker in less than five minutes. In morbid histology we often have to work rapidly. We do not agree with Dr. Colman when he says that staining operations are not easily carried out after cutting in paraffin; there is not the slightest difficulty about the matter, especially if the specimens be fixed on the cover-glasses. It is, in fact, not at all advisable to stain morbid tissues in block.

The cut paraffin sections should be floated on warm water as originally recommended by Dr. Gaskell, in order to flatten them out. We should warn neat workers against the use of bergamot oil and origanum oil; the former is redolent of a cheap hairdresser's shop, while the latter is a strong conjunctival irritant, and its odour is so strong and objectionable as to disqualify him who uses it from human intercourse. Farrant's solution may in many cases be replaced by thick cane-sugar solution, especially in mounting amyloid tissues. Sugar solution becomes quite hard, while Farrant's solution is always a nuisance, since it refuses to set. Osmic acid preparations may be mounted in Canada balsam if they are first passed through weak ammonium sulphide. There is no mention made of Weigert's modification of Gram's method, which possesses great advantages, and also stains fibrin in a brilliantly remarkable manner. Methyl blue, we wish to point out, is not the same as methylene blue.

We have looked carefully through the booklet, and have nothing more to criticise or to add; it is undoubtedly a good guide for the beginner, and therefore we have pointed out the few errors which we have detected, or suggested what seemed to us to be decided improvements. The book ought to be in the possession of all Pathology Clerks, for they would then find less difficulty in preparing good specimens, and Mr. Berry's work on Fridays would become much easier, for it is a severe task to diagnose a growth from a thick and badly stained section.—A. A. K.

Births.

CUTFIELD.—November 6th, at Merton House, Ross, Herefordshire, the wife of Arthur Cutfield, B.A., B.Sc., M.R.C.S., of a daughter.

PIERCE.—October 22nd, at The Retreat, York, the wife of Dr. Bedford Pierce, of a son.

SHARPIN.—October 12th, at 23, Kimbolton Road, Bedford, the wife of Archdale Lloyd Sharpin, of a son.

STEEDMAN.—October 28th, at Streatham, the wife of J. F. Steedman, F.R.C.S., of a daughter.

Marriage.

QUARTEY-PAPAFIO—MEYER.—On October 8th, at the Church of St. Bartholomew the Less, St. Bartholomew's Hospital, London, by the Rev. W. Ostle, Vicar, Benjamin William Quartey-Papafio, M.D.Edin., M.R.C.S.Eng., son of the late Chief Papafio, of Accra, Gold Coast, to Eliza Sabina, daughter of the late Richard S. Meyer, of Accra.

Death.

FAVELL.—On October 31st, at Brunswick House, Glossop Road, Sheffield, W. F. Favell, M.R.C.S., J.P., aged 64.

ACKNOWLEDGMENTS.—*Guy's Hospital Gazette, St. George's Hospital Gazette, St. Thomas's Hospital Gazette, St. Mary's Hospital Gazette, London Hospital Gazette, The Nursing Record, The Charity Record, The Hospital.*